ON

DEGENERATION OF THE HEART

APART FROM VALVULAR DISEASE.

Contribution to a Discussion at the Annual Meeting of the British Medical Association, Leicester, July, 1905.

ON THE

PATHOLOGY OF BRADYCARDIA.

BY

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DEGENERATION OF THE HEART

APART FROM VALVULAR DISEASE.

Dr. Hay said: I wish to confine my attention to certain changes noted in the venous pulse indicating a fundamental variation from the normal manner of the heart's contraction.

As a rule tracings taken from the jugular vein in cases of myocardial degeneration demonstrate that the inception of the cardiac rhythm is abnormal.

In the normally-contracting heart the stimulus exciting the contraction originates at the mouth of the great veins, from which point the contraction passes over the musculature spreading on to the auricular wall, then across the auricular canal, and so to the ventricle. This sequence can be observed in the tracing of a normal venous pulse (see Figs. 1, 2, 3).

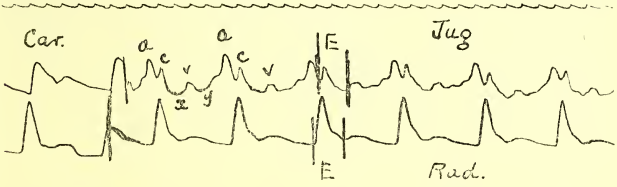

Fig. 1.—Tracing to show the auricular type of venous pulse The space E indicates in all the tracings the period of ventricular systole during which the semilunar valves are open. The wave *a*, preceding the period E, can only be due to the systole of the right auricle. The fall *x* is due to the diastole of the auricle causing a rush of blood out of the jugular vein into the auricle, and the wave *c* is due to the impact of the carotid pulse. The time marker records one-fifths of a second. This applies to all the tracings.

These three records, although somewhat dissimilar in appearance, show when analysed a similar sequence of events.

The wave *a*, due to the contraction of the auricle, occurs before the period representing the sphygmic portion of ventricular systole, namely, that portion of the tracing included between the two vertical lines, and labelled E.

The lever, after recording the wave *a*, is arrested in its fall by the impact of the carotid wave, and thus is produced the wave *c* in the tracing.

The dip *x* is due to auricular diastole, which allows the blood to pour from the jugular vein into the flaccid auricle.

The wave *v*, which ends before the auricle has begun to contract, and begins before the ventricle has ceased to contract, is caused in some way by the ventricular systole.

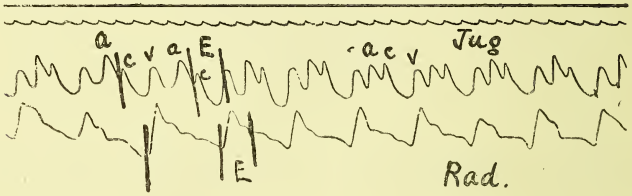

Fig. 2.—Tracing to show the auricular type of venous pulse. The form of the jugular tracing is slightly different from Fig. 1, but its interpretation is the same.

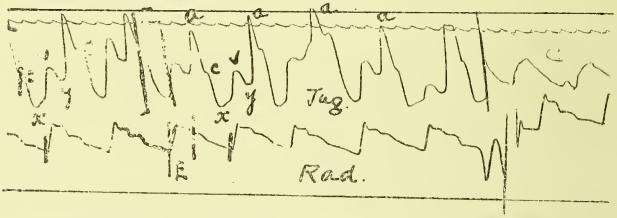

Fig. 3.—Tracing to show the auricular type of venous pulse, taken from a man of 65, with definite arterio-sclerosis of the radial artery. No palpitation and no dyspnœa. No cardiac murmurs; occasional fainting attacks.

In a large proportion of the cases of myocardial degeneration, in which the diagnosis is based upon cardiac insufficiency without evidence of valvular disease, one notes an absence of the wave *a* in the tracing obtained from the jugular vein.

This implies that the auricles are not contracting before the ventricles. The auricles are therefore either paralysed or, if not paralysed, contracting simultaneously with the ventricles. Such a venous pulse is recorded in Fig. 4.

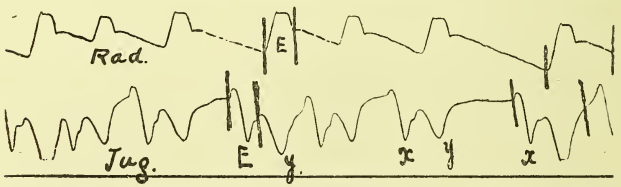

Fig. 4.—The tracing taken from the jugular vein shows the ventricular type of venous pulse. Here there is no rise in the tracing corresponding with *a* in the first three tracings; the auricle has not contracted before the systole of the ventricles. The patient, a man aged 65, suffers from slight mitral incompetence, and there is marked diminution in his "area of cardiac response." Two months ago he was at work.

If we examine this tracing, we notice that there is no sign of the wave *a* before the period E. Now, if the auricle had contracted before the ventricle, as is normally the case, the wave *a* would have been present, as in Figs. 1, 2, 3.

The inference that there was no auricular systole preceding that of the ventricle is inevitable.

In all probability the auricle and ventricle contracted simultaneously. This conclusion is based on the presence of the marked depression at *x*, which represents a sudden emptying of the veins in the neck, and can only be due to one of two causes—first, diastole of the auricle; secondly, diastole of the ventricle; as "*x*" occurs during the period E the ventricle is in systole, and so one is forced to look upon *x* in this tracing as an evidence of auricular diastole.

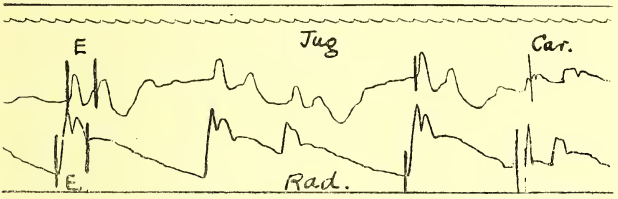

Fig. 5.—The tracing taken from the jugular vein shows the ventricular type of venous pulse. There is no sign of any contraction of the auricle preceding the period of ventricular systole, E. The heart was beating infrequently, 40 to 45 beats per minute. The patient, a man of 41, suffers from mitral stenosis and regurgitation, and there is very marked diminution in his "area of cardiac response."

Diastole of the auricle necessitates a preceding systole, therefore the wave in the tracing preceding *x* is due to auricular systole, and as it occurs during the period E the auricle and ventricle must be contracting together.

This condition of affairs is beautifully explained by Dr. Mackenzie in the BRITISH MEDICAL JOURNAL for March 5th, 1904.

Fig. 5 shows a similar condition of affairs in a patient with marked valvular disease.

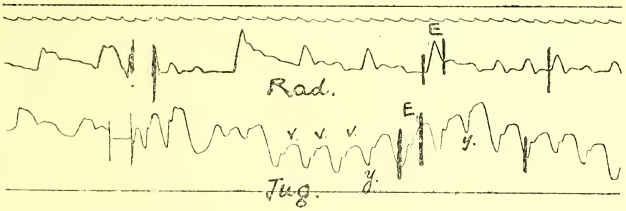

Fig. 6.—Tracing taken from the same patient as gave Fig. 5. The heart is here beating very frequently, 120 to 160 beats per minute. The jugular pulse is of the ventricular type. The radial pulse was weak and rapid, only the more forcible beats being palpable. Here either the muscular fibres of the auricular canal or those of the ventricle are responsible for the inception of the cardiac rhythm.

This patient has attacks of transient tachycardia, in which his heart beats at the rate of 160 per minute. Fig. 6 is a tracing taken during such an attack and is a beautiful example of the

ventricular rhythm. I reproduce it because of its marked resemblance to Fig. 7. And yet in the one case the patient suffered from serious mitral mischief, while in the other the valves were healthy. At the *post-mortem* examination of this latter case the pathologist described the heart muscle as soft and fatty.

The history given by this patient bears very pertinently on the present discussion. He was not conscious of any trouble till last July, when he undertook a heavy march which induced cardiac distress. From that time his pulse was frequent and irregular, and he was incapacitated for any hard work.

When I saw him with Dr. Glynn Whittle, and took this tracing, the venous pulse was ventricular in type, and I am of opinion that the sudden onset of cardiac insufficiency was due to the inception of this ventricular rhythm.

The patient referred to by Dr. Foxwell was an example of sudden onset, though, as he pointed out, there must have been myocardial changes for a long period. An examination of the venous pulse would, I believe, have demonstrated the ventricular form.

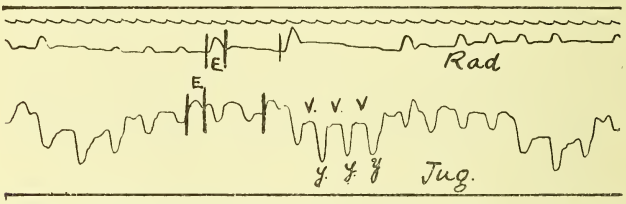

Fig. 7.—Tracing similar in every respect to that in Fig. 6. The jugular pulse is ventricular in type. Here, however, there is no organic disease of the valves. The frequent and irregular action of the heart dated from a year previous to this tracing, and was caused by a forced march. The autopsy revealed normal valves, some incompetence of the mitral and tricuspid orifices, due to dilatation of the ventricles, and a soft fatty myocardium.

Myocardial degeneration is not produced in a few hours. It had probably existed here for months and years without giving rise to any appreciable discomfort.

That the auricles and ventricles can, under certain circumstances, beat simultaneously has been proved experimentally by Lohmann.[1] He initiated the condition by the application of electrical stimuli to the auricular canal at the site of Gaskell's bridge. On the cessation of the electrical stimulation the abnormal rhythm continued; the stimuli originating in the auricular canal and passing simultaneously upwards to the auricle and downwards to the ventricle.

As Gaskell[2] has pointed out, the more embryonic portions of the heart, such as the fibres at the junction of the sinus and the auricle, and the fibres in the auricular canal, possess certain characteristics different from those of the more developed portions, one characteristic being that of a much greater rhythmicity or power to develop a rhythmical beat. If, therefore, from any cause the circular fibres at the junction of the large veins with the auricle fail to initiate the

[1] Zur Automatie der Brückenfasern und der Ventrikel des Herzens. *Archiv für Anat und Physiologie*, 1904, p. 431.

[2] The Contraction of Cardiac Muscle. *Textbook of Physiology*. Edited by Schäfer, vol. ii.

contraction, and so fail to dominate the rhythm, there are still the embryonic fibres of the "auricular canal" possessing this power in a marked degree.

Last year Dr. Mackenzie, in the valuable paper already referred to, gave several instances in which the inception of the rhythm proceeded from the ventricle, and he also gave a detailed account of a most instructive case where the auricles and ventricles undoubtedly contracted simultaneously. In such cases one finds that the heart's action is practically always irregular, and that the abnormal rhythm tends to persist.

When the pulse is very frequent, the action is more regular, the irregularity reappearing as the heart slows down (see Figs. 5, 6, and 7). In my opinion many patients in whom the diagnosis of myocardial degeneration is made are in reality cases in which the muscular fibres in or near the auricular canal have usurped the domination of the rhythm at first intermittently, then sooner or later permanently.

As a rule, one does not see the patient until the abnormal inception of the rhythm is established, and then the dyspnoea, the irregular pulse, the dilatation of the heart, the altered venous pulse, and the diminished area of cardiac response are all ascribed to the supposed changes in the myocardium.

One must not forget that all these signs and symptoms of cardiac insufficiency do sometimes occur in hearts where there

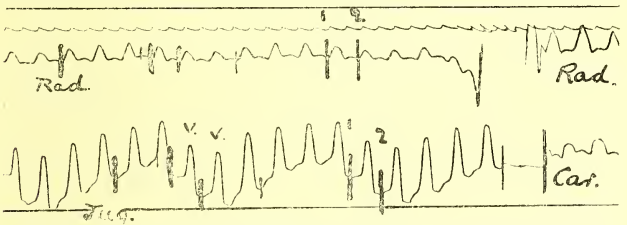

Fig. 8.—Tracing taken on February 23rd, 1905, from a child of 6½ years suffering from "paroxysmal tachycardia." Heart frequency of 190. The venous pulse is of the ventricular type; there is no sign of any contraction of the auricle. There is nothing in the history of the child to suggest the presence of any cardiac degeneration. Owing to the tachycardia the cardiac dullness is increased to the right and to the left; the heart is dilated.

is no reason to suspect any lesion of the myocardium or of the valves. Such happens undoubtedly in cases of paroxysmal tachycardia, where a heart normal in its past history, in its size, in activity, and in area of cardiac response, suddenly bursts into a frequency of 160 to 200 beats per minute. In a few hours we find that heart dilated, its area of response diminished, possibly signs of visceral congestion, and, most important of all, that the auricle has ceased to contract before the ventricle.

This is well shown in Fig. 8, a tracing taken from a small boy of 6½ years suffering from an attack of paroxysmal tachycardia. The tracing from the jugular vein shows no sign of auricular contraction. There is only one wave, and that is ventricular. The heart was beating at 190 per minute; it was dilated, and the boy suffered from dyspnoea on exertion.

In this case the attack did not cease suddenly, and one is able to note in the tracings of the venous pulse the gradual

reappearance of the normal auricular contraction (see Figs. 9 and 10).

In Fig. 9 the normal inception of the rhythm by the auricle occurs after every two or more beats.

In Fig. 10 one finds every second, third, or fourth contraction is dominated by the auricle.

Fig. 11 shows a tracing taken immediately after the attack; the radial pulse is regular, the venous pulse has returned wholly to the auricular type, every systole of the ventricle being dominated by the auricle. The heart rapidly recovered its normal size and tone, and the child is now healthy and strong.

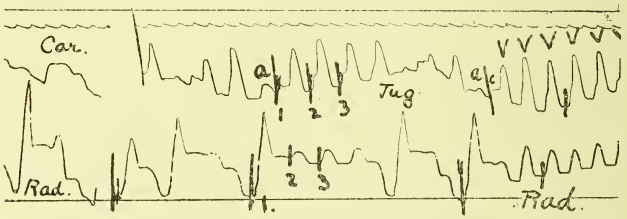

Fig. 9.—Tracing from the same patient as gave Fig. 8. Taken March 6th, 1905. Note that the ventricular rhythm is not continuous, the pulse is irregular. There is a large beat followed by several small ones. On analysing the venous pulse tracing one finds that whenever there is a full beat in the radial artery there is a wave in the jugular vein due to the contraction of the auricle preceding that of the ventricle.

I have referred to this case in order to emphasize two points, first, that the ventricular rhythm can occur in a normal heart; secondly, that the inception of the ventricular rhythm, even in a normal heart, can, and does, give rise to all those signs of cardiac insufficiency which in the absence of valvular disease are said to justify a diagnosis of myocardial degeneration.

Through the kindness of Dr. Mackenzie I had an opportunity of seeing and examining a woman, aged 57, in whom this ventricular rhythm was present, and persistent. There was marked cardiac insufficiency, but no valvular disease.

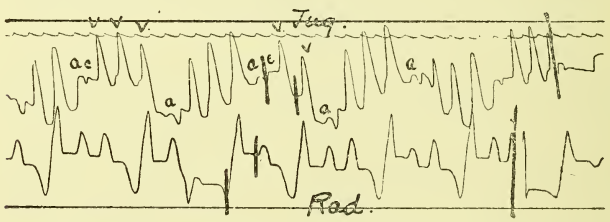

Fig. 10.—Another tracing taken from the same patient, March 6th, 1905, showing the transition stage from the ventricular to the auricular type of venous pulse. When the auricle dominates the rhythm there is a full and strong response on the part of the ventricle, otherwise the contraction is weak and ineffective.

She has since died, and this latter statement corroborated —myocardial changes were found *post mortem*.

He had observed the onset of this condition of ventricular rhythm in an apparently normal heart, the first variation from

the normal being attacks of a frequent, irregular pulse; during these attacks of irregularity the rhythm was ventricular, between the attacks the cardiac action was normal. *Here the cardiac distress and disability were not the cause of the ventricular rhythm, but were secondary to its inception. The cardiac action cannot be other than inefficient when the stimulus originates in any site other than the normal.*

This is well illustrated in Figs. 9 and 10, for one sees in these tracings that whenever the auricular systole precedes the ventricular, that the action of the heart is strong and effective, and a good full wave is sent to the periphery; on the contrary, when one sees from the tracing that the auricle has not contracted before the ventricle, the contraction of the heart is feeble and unable to send a powerful pulse to the radial artery. When the auricle dominates the rhythm of the heart, the action is markedly more efficient than when the ventricle or auricular canal take on the inception of the rhythm, and this quite irrespective of myocardial degeneration.

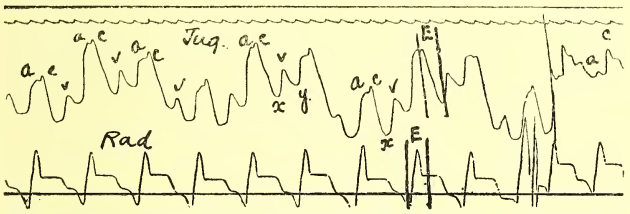

Fig. 11.—Tracing from the same patient, March 15th, 1905. The pulse is regular with a frequency of about 80 per minute. The venous pulse is of the auricular type and the cardiac dullness normal.

It may and probably will be suggested that this form of rhythm, in which there is no contraction of the auricle preceding that of the ventricle, is due to changes in the cardiac musculature, and that the degeneration of the cardiac fibres is always the primary factor.

If it had not been for these peculiar and interesting cases of paroxysmal tachycardia and such cases as that of Dr. Mackenzie's, with others of similar nature, one would have felt inclined to accept the above view as probably correct; but, as Lohmann has demonstrated experimentally, and as we have seen in the patient suffering from tachycardia briefly described above, this ventricular rhythm does occur in hearts where there is no reason to suspect any degeneration of the musculature. We must therefore admit the possibility of the inception of this form of rhythm apart from any myocardial degeneration, and also that the onset of this abnormal rhythm is sufficient in itself to produce cardiac distress and disability.

Why the heart should take on this abnormal rhythm I cannot say. It must be due to a depression of the musculature at the mouth of the large veins as they open into the auricle—a "negative chronotropic condition," in the language of Engelmann and Wenckebach—or to an exalted condition of the auricular canal and surrounding cardiac muscle.

What the factors are which produce changes in the excitability of these parts is not known, though it is quite conceivable that changes in the nutrition of the myocardium may act in this manner. What I would specially like to empha-

size is that the onset of marked symptoms is largely independent of the presence of the myocardial degeneration, and is synchronous with the onset of the abnormal inception of the rhythm.

The cardiac insufficiency is due to this factor rather than to any associated myocardial disease, and even though the *post-mortem* examination reveals some signs of myocardial degeneration, still the heart owed its inefficiency not to the degeneration, but to the abnormal inception of the rhythm.

To summarize, in conclusion:

1. The venous pulse is of the ventricular type in the majority of those cases of so-called "chronic myocarditis" in which there are signs of cardiac disability—namely, palpitation, dyspnoea, irregular pulse, and diminution of the area of cardiac response.

2. This abnormal inception of the cardiac rhythm, as demonstrated by the ventricular type of the venous pulse, can and does frequently occur irrespective of myocardial degeneration. The paroxysmal tachycardia met with in the boy aged $6\frac{1}{2}$ years is a case in point, where there are no grounds for assuming the existence of any myocardial degeneration. The dilatation of the heart, the cardiac distress, the irregular action, and the diminished area of cardiac response were all secondary to the onset of the tachycardia, and disappeared almost as suddenly as they had appeared on the heart resuming its normal mode of action.

3. The conditions are not known which alter the excitability of the musculature of the auricular canal and adjoining portions of the ventricle. It is probable that changes in the nutrition of the myocardium act in this manner.

4. The onset of marked symptoms is largely independent of the myocardial degeneration, but is synchronous with the onset of the abnormal inception of the rhythm.

5. This rhythm, with its consequences, sometimes occurs in the absence of any evidence of myocardial degeneration.

6. The heart's action is always inefficient in the presence of the ventricular rhythm, whether myocardial changes be present or not.

THE PATHOLOGY OF BRADYCARDIA.

THERE is a form of bradycardia termed "paroxysmal" or "intermittent." Two varieties are described: (1) In which the pulse is permanently infrequent, and in which there are sudden accesses of greatly-diminished frequency; (2) where the pulse is of normal frequency between the attacks.

SUMMARY OF CASE.

The case which I shall now relate belongs to the first variety. The following are the important points to be noted in reference to it:

(*a*) Marked bradycardia, the pulse frequency ranging from 17 to 30 per minute, "à pouls lent permanent."

(*b*) Transient loss of consciousness, associated with cessation of the pulse at the wrist.

(*c*) Auricular contractions occurring twice or thrice as frequently as the ventricular, the cause of the bradycardia being "heart block."

(*d*) An enormous heart, the autopsy revealing no cause for the hypertrophy and dilatation.

I give a brief summary of the case:

R. M., aged 26, was healthy till May, 1898, when he suddenly lost consciousness. Some time later he was admitted to the Royal Infirmary under the care of Dr. Barr, to whom with Dr. Raw I am much indebted for permission to publish this case. On admission he complained of "breathlessness on exertion and attacks of unconsciousness." On January 8th, 1900, he entered Mill Road Infirmary suffering from extreme cardiac failure. He died on January 15th, 1900.

When he was in the Royal Infirmary I noted the following points:

1. The apex beat was situated well outside the nipple line, slow, heaving, and forcible in character, 20 beats per minute.

2. Pulsations or small beats, numbering 50 or 60 per minute, occurred regularly over the third left interspace, just to the left of the sternum.

3. Synchronous pulsations were observed in the veins of the neck.

4. Definite systolic retraction in the third, fourth, and fifth left spaces.

5. Cardiac dullness (deep) extended 7 in. to the left, 2 in. to the right of the middle line.

6. A systolic murmur at all areas, loudest and harshest over the conus of the right ventricle. It was conducted to the left and well heard along the vertebral border of the scapula.

7. No sounds were audible accompanying the small pulsations mentioned above.

8. Pulse varied from 17 to 30 per minute.

Fig. 1 is a pulse tracing. A cardiogram was taken simultaneously with a record of the venous pulse in the neck. The

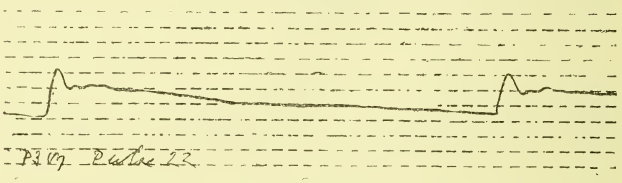

Fig. 1.—Pulse tracing taken by Dr. Barr. Rate, 20 per minute.

diagram (Fig. 2) is an accurate representation of the tracings. The cardiogram demonstrates the slow rhythm of the ventricle and the more frequent rhythm of the auricles. When the contraction of the auricles coincides with that of the ventricles it is masked by the record of the ventricular

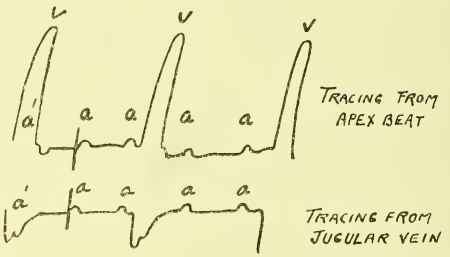

Fig. 2.—v = wave caused by ventricular systole; a = wave caused by auricular systole. At a' the auricle contracted, but its record is masked by the larger record of the ventricular systole, which occurred at the same moment.

contraction. The waves in the venous pulse tracing are synchronous with those of the cardiogram. The tracings show, therefore, the two independent rhythms, auricular and ventricular.

The pulsations visible over the conus of the right ventricle were due, in my opinion, to the strong right auricle forcing blood through into the right ventricle, the ventricle being in diastole and permitting the impact of blood to make itself evident by a slight movement of the chest wall over the conus.

Later, when admitted to Mill Road, it was noticed that the cardiac dullness (deep) had increased to the right by 1 in., that the only murmurs persisting were a marked tricuspid regurgitant and a faint mitral regurgitant, and that the frequency of the pulse had reached 40 to 80 per minute.

Necropsy.

The necropsy, to my astonishment, gave little assistance. There was some cyanosis of the lips and ears, slight general jaundice, and a little puffiness about the ankles. The chart of the deep cardiac dullness, dated January 14th, was mapped out on the chest, and long needles were inserted at right angles to the surface. To the right the needle was found to have entered the pericardial sac without touching the heart; at the apex the needle just entered the edge of the left ventricle, thus demonstrating that the deep cardiac dullness as elicited *ante mortem* was correct.

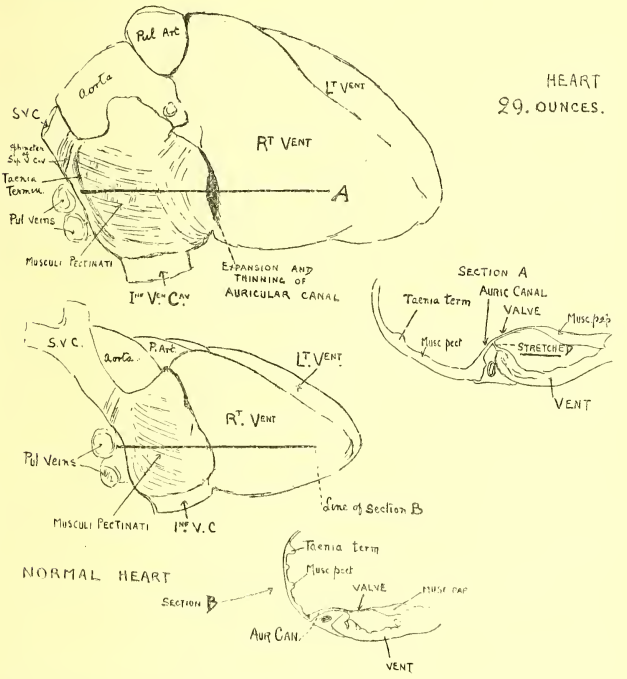

Fig. 3.—Diagram drawn by Professor Keith. It compares the heart of the patient with a normal heart, and demonstrates the condition present at the auricular canal. (Drawn to scale.)

The pericardium was normal, and contained 1 oz. of clear fluid. The heart weighed 29 oz., and showed marked hypertrophy and dilatation. The valves were all translucent, and of a normal appearance. The tricuspid and mitral valves were incompetent. The lungs were tough and doughy, and comparatively pale and dry. There were old pleural adhesions, and there were two pulmonary apoplexies in the right lower lobe. The liver was engorged and nutmeg. The kidneys weighed 15 oz., and on microscopical examination were found to be normal, except for signs of back pressure. There was no evidence of arterio-sclerosis.

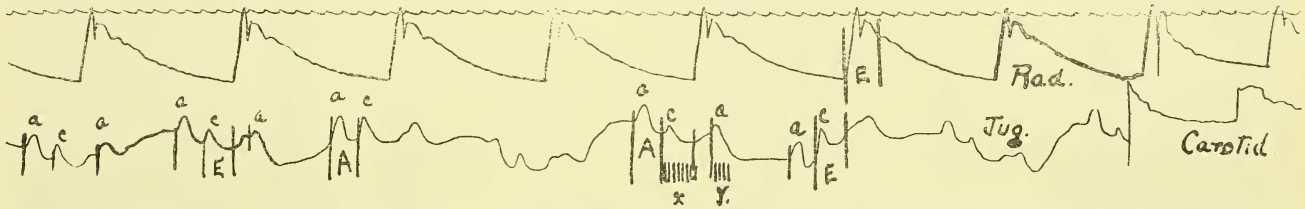

Fig. 4.—Simultaneous tracings of the radial and jugular pulses, showing that the *a-c* interval (space A) is twice the normal period (two-fifths of a second in place of one-fifth), indicating a delay in the passage of the stimulus from auricle to ventricle. It shows also that only every other systole of the auricle is followed by a systole of the ventricles. *x* = murmur due to ventricular systole, mitral regurgitation ; *y* = murmur produced by auricular systole discharging blood into an almost empty ventricle. The time marking here represents one-fifth second. The space E indicates the period of ventricular systole during which the semilunar valves are open—the sphygmic period.

Some weeks ago I sent the heart to Professor Arthur Keith, of the London Hospital, for his opinion. With exceptional kindness he has drawn a diagram showing its condition, and more particularly that of the auriculo-ventricular junction. His method of examining hearts may be as new to others as it was to me. I therefore reproduce the diagram (Fig. 3). From his report I quote the following :

"The two marked changes in your specimen (besides the hypertrophy and dilatation of the chambers) are: (1) Stretching of the auricular canal (the part normally within the auriculo-ventricular groove); (2) separation, or rather attenuation, of the bond between the bases of the valves (tricuspid) and base of the ventricle." These points are well shown in the diagram.

Remarks.

Here is an undoubted case of "heart block." Normally the stimulus to contraction is initiated in the remains of the sinus venosus, consisting of the musculature at the mouth of the great veins and adjoining portion of the right auricle. The wave of contraction spreads over the auricle and through the auricular canal on to the ventricle "with varying speed, quickest over the tissue which has become modified so as to approach more nearly in its properties to ordinary striated muscle—namely, the reticulated bulged portion of the auricle and ventricle; more slowly over those parts which retain a more embryonic character—namely, the auriculo-ventricular muscular ring and the bulbus arteriosus."[1]

Normally the ventricle responds to a stimulus spreading down to it from the auricle; if by any means the ventricle is cut off from this normal periodic stimulus it tends to initiate its own stimulus and to take on a rhythm different from and independent of the auricle. Gaskell demonstrated this in the frog's heart by ligaturing the auriculo-ventricular groove so that no stimulus could cross from auricle to ventricle. The term "heart block" is applied to this condition.

A heart in which the auricle dominates the rhythm as a rule beats with normal frequency. When the stimulus for ventricular action arises in the ventricle itself there is a slow rhythm. The result of "heart block" is therefore a bradycardia. Such a condition was present in this case; the block in all probability occurred at the attenuated portion of the ventricular wall situated at the base of the ventricle at its junction with the auricular canal.

An interesting question arises as to the cause of the great hypertrophy and dilatation of the heart. It is too great to be accounted for satisfactorily by the strain of work. It was not due to chronic nephritis; there was no sclerosis of the valves. The incompetency of the mitral and tricuspid must be in some degree responsible, but this pertains rather to the later stages than to the period of comparative compensation, when, as I have already noted, there was a transverse cardiac dullness of 9 in. I venture the following suggestion :

Since during each ventricular diastole the auricles contract twice or thrice, the resulting condition of the ventricles would be one of over-distension, and they would during their systole have an excessive amount of blood to deal with. The strain on the ventricular walls due to the forcible pumping in of blood by the auricles would produce dilatation, and the large volume of blood dealt with would necessitate hypertrophy.

The discussion of this case would be incomplete if it did not include a brief reference to the "attacks of unconsciousness"

of which the patient complained. Twice on March 23rd, 1899, it was noted that during an attack of dizziness his pulse became imperceptible, returning gradually to its normal rate of 20 a minute. I believe that this explains the loss of unconsciousness, and brings this case into line with the more pronounced forms of the "Adams-Stokes syndrome."

In this case such attacks were infrequent and not at all pronounced; convulsions in relation to them were not noted at any time, but, with the notes before us, I think I am justified in assuming that the dizziness and fainting were due to temporary cessation of ventricular contraction.

A case, somewhat similar but much more marked, is recorded by A. Webster.[2] His patient complained of taking what he called "weak turns associated with palpitation, sensations of giddiness, and occasional loss of consciousness." The record is illustrated by a series of most beautiful tracings demonstrating the disappearance of the radial pulse coincident with the dizziness and loss of consciousness.

We may take it, then, that in the case I have described the bradycardia was due to a condition of heart block so complete that the auricles and ventricles beat independently of each other.

There are other cases, however, in which the block in the auricular canal is not complete, and where the ventricles respond to every second contraction of the auricles, or even to every third or fourth contraction. Quite recently I met with such a case in which the bradycardia was caused by a severe depression of conductivity, the ventricle failing to respond to every second contraction of the auricle. Two similar cases are described and figured by Mackenzie[3] in his recent excellent paper in the BRITISH MEDICAL JOURNAL of March 11th, 1905.

The patient I refer to is 62 years of age. His occupation up to the present illness was that of a baker. This entailed the carrying of heavy loads up and down stairs. He denies syphilis or alcoholic excess.

The present illness began three years ago with a sudden loss of consciousness. Since then he has occasionally suffered from "mazey bouts." The doctor in attendance tells me that when he first saw him eighteen months ago he noticed that the pulse was 20 a minute; there was then some bronchitis, with considerable dyspnoea; the extremities were cold, but there was no cyanosis or oedema.

Since that time his pulse has remained persistently infrequent, and the bradycardia probably dates from then.

I was surprised by the healthy appearance presented by the man. He had a good colour, and was well nourished, but I noticed signs of deficient aëration of the blood in the hands, which were very cold, and, according to the patient, became livid and numb on the slightest exposure. He felt perfectly well with the exception of great dyspnoea on exertion. The "area of cardiac response" was very limited. There was marked emphysema of the chest. The radial and temporal arteries were uniformly thickened and tortuous. The pulse was 32 to the minute, regular in force and frequency, of large volume and good tension. I noticed two beats in the neck to each radial pulse, and took tracings to demonstrate this (Fig. 4).

On analysing these records it will be seen that the waves due to auricular systole occur at regular intervals, and that every other such wave is followed by the carotid pulse. It is almost impossible to record the venous pulse in the neck without at the same time recording the carotid. This is

rather an advantage than otherwise, as it gives a fixed point in the analysis of the tracing. In the record of the jugular pulse the distance from the beginning of the contraction of the auricle to the first appearance of the carotid beat corresponds roughly to the time between the auricular and ventricular contraction. This interval is called the *a–c* interval, and is normally about one-fifth of a second in duration. Where, however, there is depression of conductivity, and the stimulus finds greater difficulty in passing from auricle to ventricle, this *a–c* interval is lengthened. It is so here; instead of being one-fifth of a second it is two-fifths of a second. An examination of the tracing shows, first, an auricular contraction *a*, followed by the carotid pulse *c*, and that the *a–c* interval is double the normal duration; then we meet with another wave *a*, due to a second contraction of the auricle, but this time there is no sign of a ventricular response. The stimulus has not been able to reach the ventricle. This sequence is repeated without variation in all the tracings.

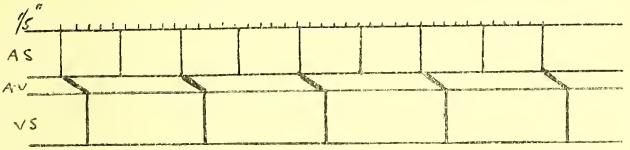

Fig. 5.—Diagram constructed to show the action of the heart when Fig. 4 was taken.

To demonstrate the action of the heart when Fig. 4 was taken I have constructed the diagram Fig. 5. It is built on the plan adopted by Wenckebach and Mackenzie. The down strokes in the upper division, A S, represent the auricular systoles and correspond with the waves "*a*" in Fig. 4, while the down strokes in the lower division, V S, represent the ventricular systoles, and correspond to the radial and carotid pulses in Fig. 4. The short, thick lines connecting these down strokes represent the passage of the stimulus through the fibres joining auricle and ventricle. In this case they are two-fifths of a second in duration—that is, double the normal length. The absence of this line indicates that the stimulus has failed to pass these fibres, and hence a ventricular systole drops out.

I also obtained a tracing combining a cardiogram and a sphygmogram, which demonstrates that in this case every ventricular contraction is represented by a pulse wave in the peripheral arteries. The heart is little, if at all, enlarged, the deep cardiac dullness measuring $1\frac{1}{2}$ in. to the right and 4 in. to the left of the middle line. On auscultation I found that it was difficult to hear the heart sounds at the base owing to the emphysema. At the apex I made out a wheezy systolic bruit, followed by a clear second sound, and then during the diastole of the ventricle, and synchronous with the extra pulsation in the neck, I heard a short, whiffing murmur, due, I take it, to the auricular systole forcing blood into the partially-filled ventricles.

REFERENCES.

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